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PROCEEDINGS
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THE TIME FACTOR IN X-RAY PRODUCTION OF CHROMOSOME ABERRATIONS

By Karl Sax

Arnold Arboretum, Harvard University

Read before the Academy, April 24, 1939

Cells exposed to x-rays are affected in various ways. The first apparent response is a temporary inhibition of nuclear division. The cells already in the process of division continue to divide, but the chromosomes are clumped and appear to be sticky. Changes in the permeability of the protoplasmic membranes may occur, and the viscosity of the cytoplasm may be altered. Recent evidence indicates that the activity of enzymes and hormones may be inhibited. At low concentrations of irradiation these effects appear to be temporary and the cell recovers. The more permanent effects involve the changes induced in the chromosomes. These changes are of two types, structural alterations in the chromosomes and gene mutations. Either type of chromosome change may lead to variation in the organism, or may be lethal at some point in the cell lineage.

The chromosome aberrations to be described were induced in the developing microspores of Tradescantia. The microspore nucleus remains in the resting stage for five or six days. During this time the chromosomes are single threads in the form of relic spirals. The nucleus then enters the prophase stage and each chromosome becomes split lengthwise to form two sister chromatids. This splitting of the chromosomes begins about thirty hours before metaphase and is completed in all chromosomes at about twenty-four hours before metaphase. Thus the nuclear cycle, from the tetrad stage following neiosis to the metaphase stage in the division of the microspore nucleus, requires about a week. This cycle is somewhat longer in the winter months. Division figures examined 24 hours after irradiation were rayed at prophase, while those fixed five days after raying were in the resting stage when x-rayed.

Two general types of chromosome aberrations are induced by x-rays—chromatid breaks and chromosome breaks. The chromatid breaks are

induced at prophase when all chromosomes consist of sister chromatids. Either one or both of the sister chromatids may be broken by a single hit, i.e., the ionization produced by a single quanta. If both chromatids are broken, the broken ends of adjacent sister chromatids may fuse to produce a dicentric chromatid and a U-shaped fragment. When chromatid breaks are induced in several chromosomes, fusions may occur between broken ends of different chromosomes and produce chromatid bridges, translocations, or rings. These aberrations are dependent upon two independent hits. The frequency of the one-hit chromatid breaks is approximately proportional to dosage $(D/K)^{1.2}$, while the two-hit chromatid aberrations show an exponential increase with increased dosage $(D/K)^{2.0}$.

Irradiation of the resting nucleus produces chromosome breaks. Single hits are rarely effective in producing aberrations at this stage because the chromosome consists of a single thread. A single hit produces only two broken ends at this time and the two broken ends usually reunite in the original position so that no aberration appears at metaphase. Most of the chromosome aberrations are produced by fusions between broken ends of different chromosomes or chromosome arms. These dicentric, reciprocal interchange and ring chromosomes are dependent upon two independent hits, and show the same relation to dosage as the two-hit chromatid aberrations (Sax,[1] and unpublished). With the exception of reciprocal interchanges, all aberrations are accompanied by chromosome or chromatid fragments. These acentric fragments are fused terminal deletions. They are rarely included in the daughter nuclei, and all are lethal in the haploid cell lineage.

If chromosome aberrations are dependent upon the occurrence of two breaks within certain limits of time and space, the frequency of such aberrations should be dependent upon the intensity of the radiation. If broken ends of chromosomes fuse quickly, a low intensity should produce few aberrations because a single break would be re-fused before a second break occurred in an adjacent chromosome. But if broken ends remain in an unstable condition for a comparatively long time and fuse with other broken ends only when they happen to come in contact, a given dosage should be equally effective at either high or low intensity.

The relation between the chromosome aberrations and the time factor can be determined by giving a series of flower buds the same x-ray dose, but at different intensities, or by raying the flower buds at the same intensity, but with varying intermittent exposures. In each of these experiments we have used buds from a single plant or a clonal line. The microspores were fixed and stained 24–26 hours after raying for the analysis of chromatid breaks and five days after raying for a study of chromosome breaks. Only dicentric and ring chromosomes were scored in listing the chromosome aberrations because terminal deletions are rare and reciprocal

interchanges can be recognized only when the exchange is very unequal. The two-hit chromosome and chromatid aberrations involve two breaks and are so classified in obtaining the percentage of breaks.

TABLE 1

THE EFFECT OF THE TIME FACTOR IN X-RAY INDUCED CHROMOSOME ABERRATIONS. 134 K. V. 10 MA. 320 R. 5 day

DURATION OF RAYING	R/M	TOTAL CHROMOSOMES	EXCHANGE BREAKS	% BREAKS
1′	320	2844	492	17.3
2′	160	1044	140	13.4
4′	80	1878	218	11.6
8′	40	2262	206	9.1
16′	20	2592	182	7.0

In the first experiments on the time-intensity factor each series was set up so that all flower buds received the same x-ray dose, but at different intensities. The intensity of radiation was varied by placing the flower buds at varying distances from the x-ray tube following the inverse square

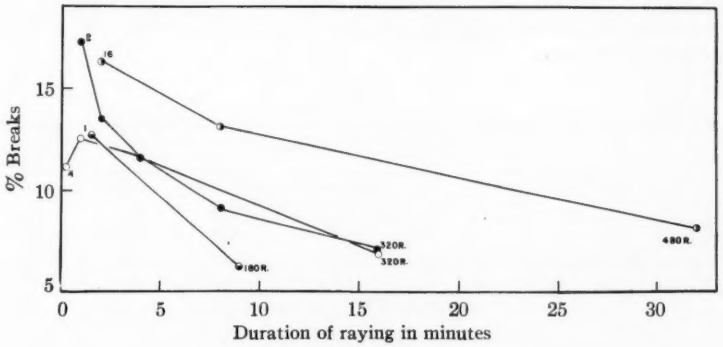

FIGURE 1

The relation between the time-intensity factor and frequency of x-ray induced chromosome aberrations.

law. The time of exposure was varied for each intensity so that all buds received the same x-ray dosage. The results of one of the first experiments are shown in table 1. The buds rayed for one minute at 320 r/m show more than twice as many chromosome aberrations as those rayed for 16 minutes at 20 r/m. The results are shown graphically in curve 2 of figure 1. The relation between radiation intensity and frequency of chromosome breaks is expressed by the equation $\log \%\ \text{breaks} = 0.314 \log I + 0.764$, where I is the radiation intensity in r/m. Obviously this curve cannot be extrapolated indefinitely in either direction, and we do not have the neces-

sary data to determine the intensity which should practically eliminate chromosome aberrations. It must be quite low, however, perhaps several roentgens per hour.

These experiments on the time-intensity factor were repeated at various x-ray intensities and at different total doses. The results of these experiments are shown in the form of curves in figure 1. The data in experiment 1 were obtained from an analysis of only 2208 chromosomes, but number 4 is based on an analysis of 14,040 chromosomes, and 5070 chromosomes were examined in experiment 16. The results shown in experiment 4 are not consistent, due to the difficulties in calibration at a short distance from the tube (15 cm.) and possible errors in timing the exposure at 15 seconds. In this experiment the voltage was 200 K. V. at 25 ma. with filters equivalent to 0.25 mm. of copper and 2 mm. of aluminum. All other experiments were done at 134 K. V. 10 ma. without filters. The tube distance varied from 19 to 100 cm.

In all cases there is a decrease in the frequency of chromosome aberrations with longer exposure and decreased x-ray intensity. There is some correlation between the slopes of the curves and the total dosage. The frequencies of chromosome aberrations decline more rapidly with time at the lower intensities.

TABLE 2

THE EFFECT OF INTERMITTENT RADIATION ON INDUCED CHROMOSOME ABERRATIONS. 134 K. V. 10 MA. 25 R/M. 5 DAY

	EXPOSURES NO.	EXPOSURES TIME	REST PERIODS NO.	REST PERIODS DURATION	TOTAL CHROMOSOMES	EXCHANGE BREAKS	% BREAKS
1	1	16′	0		5388	886	16.4
2	4	4′	3	16′	3816	514	13.5
3	4	4′	3	32′	5820	582	10.0
4	4	4′	3	48′	4014	286	7.1
5	1	4′	0		7440	98	1.3

The relation between frequency of chromosome aberrations and x-ray intensity shows that many of the chromosome breaks reunite in the original position very soon after they are induced by irradiation, but we have no precise information on the maximum time a break can remain "open" in an unstable condition. This period can be determined from intermittent timing experiments. Four experiments were completed using x-ray intensities of 12.5, 25, 50 and 160 r/m, respectively. The results of experiment 2 are shown in table 2. In each of the five series of exposures the x-ray intensity was 25 r/m. The first series was given continuous radiating for 16 minutes, while series 2, 3 and 4 were given split doses of 4 minutes each, with rest periods between exposures of 16, 32 and 48 minutes, respectively. Series 5 was given only one 4-minute exposure. The percentage of breaks induced by this single exposure, multiplied by 4, should give

the end-point or base line for comparison with the intermittent exposure series. The point at which the curves reach the base line shows the time during which a break can remain in an unstable condition. In experiment 2 the base line is 5.2 per cent. The percentage of aberrations is 16.4 for continuous radiation and decreases with increasing rest periods, but does not reach the base line with 48-minute rest periods. By projecting the curve it appears that breaks may remain open for about one hour. The results of this and other intermittent timing experiments are shown in figure 2. Curve 1 is based on four 4-minute exposures, with rest periods of 0, 4 and 16 minutes. The total number of chromosomes examined was

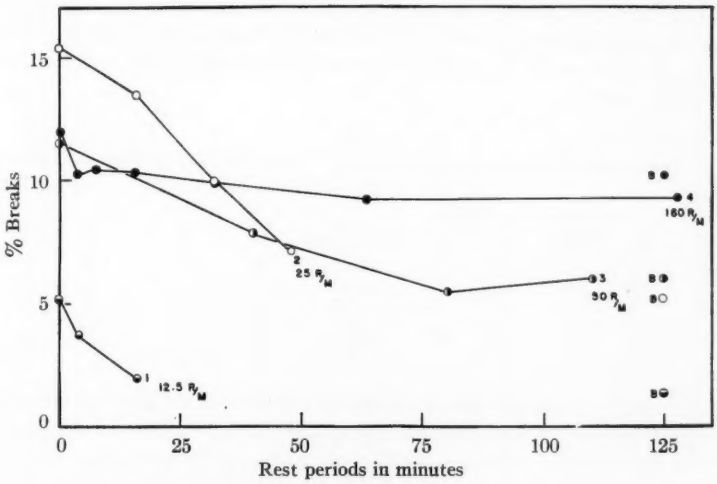

FIGURE 2

The relation between intermittent exposures to x-ray and the frequency of chromosome aberrations.

7482. Curve 3 is based on four 2-minute exposures, with rest periods of 0, 40, 80 and 120 minutes. The number of chromosomes analyzed was 19,956. Curve 4 is based on two 1-minute exposures with rest periods of 0, 4, 8, 16, 32, 64 and 128 minutes with a total of 30,282 chromosomes examined. In all cases the base lines were determined. There is considerable correlation between the radiation intensity and the slopes of the curves. At 12.5 r/m the base line is reached at about 25 minutes while at 25 r/m and 50 r/m the base line is reached with rest intervals of about one hour. At high intensity, 160 r/m, there is little or no effect of the single rest periods. The rather consistent lowering of the curve below the base line in both experiments 3 and 4 may be due to a physiological dis-

turbance caused by radiation, although the individual deviations are not statistically significant. It appears from these data that chromosome breaks can remain open in an unstable condition and are capable of fusion with other broken chromosomes for somewhat more than an hour. The nuclear cycle from meiotic telophase to the division of the microspore nucleus requires more than a week during the winter months.

The results of the various differential and intermittent timing experiments can be attributed to the fact that a chromosome aberration is produced by two independent x-ray hits. If the radiation is given at high intensity there is a greater chance for the production of adjacent breaks before the broken ends reunite in the original position. But if the radiation is delivered slowly, the broken ends of one chromosome may reunite before a second break occurs in an adjacent chromosome. No aberration is produced because the broken ends of both chromosomes reunite in the original position.

TABLE 3

THE EFFECT OF INTERMITTENT RADIATION ON 1-HIT AND 2-HIT CHROMATID BREAKS. 134 K. V. 10 MA. 25 R/M 24 HR.

	EXPOSURES		REST PERIODS			1-HIT BREAKS		2-HIT BREAKS	
	NO.	DURATION	NO.	DURATION	TOTAL CHROMOSOMES	NO.	%	NO.	%
1	1	6	0		3864	127	3.3	138	3.6
2	3	2	2	3′	2736	76	2.8	88	3.2
3	3	2	2	9′	2628	88	3.3	54	2.1
4	3	2	2	27′	1890	55	2.9	30	1.6
5	1	2	0		3948	39	1.0	24	0.6

According to this interpretation the time factor should have an influence on the frequency of 2-hit breaks, but the 1-hit breaks should be independent of the time factor. This assumption can be tested by comparing the 1-hit and 2-hit breaks induced at prophase. The results of this experiment are shown in table 3. Continuous radiation was given for six minutes and the intermittent treatments were given three 2-minute exposures, each with rest periods of 3, 9 and 27 minutes, respectively. The single exposure of two minutes provides data for determining the base line where longer rest periods are not effective in reducing the frequency of aberrations. It is evident from the results shown in table 3 that the 1-hit aberrations are independent of the time factor, but the 2-hit aberrations decrease in frequency with longer rest periods. The data are shown in graphic form in figure 3. The curve for the 2-hit breaks reaches the base line at about the 20-minute rest interval.

The results of the experiments on the time factor in relation to frequency of chromosome aberrations seem to be in accord with previous work on the time-intensity factor in relation to lethal or injurious effects on tissues and organisms. In a review of the literature Pack and Quimby[2] summarize

the results of 34 investigators who have studied the time-intensity factor in radiation experiments. The results in nine of these experiments showed no effect of the time-intensity factor; five investigators found a greater effect at lower intensities, while 20 of the experiments showed that the high intensities of radiation were more injurious. Some of the discrepancies can be explained on the basis of differences in the duration of the nuclear cycle in different materials. If the cell cycle is rapid and the radiation intensity is relatively low, injury occurs only at the most sensitive period, i.e., at the time of division, and long exposures of low intensity will be more

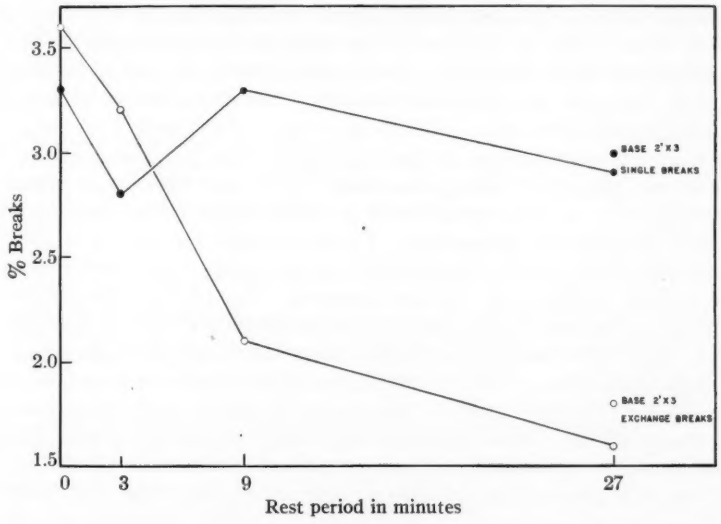

FIGURE 3

The effect of intermittent x-ray radiation on 1-hit and 2-hit chromatid aberrations.

effective than short exposures of higher intensity. The short exposure will not be so effective because few of the cells will be at the sensitive stage during the period of raying. With most cells the nuclear cycle is longer than the longest exposure time, so that most of the nuclei are in similar stages of development. Under such conditions a high intensity of radiation is more effective than the same dose given at lower intensity, because most of the nuclei are in the resting stage where two hits, limited to a critical time period, are necessary to produce a chromosome aberration. The time-intensity factor shows no effect on the mutation rate, because most mutations induced by x-rays are caused by single hits.

It has been assumed that chromosome deletions can persist as detached

fragments for several cell generations and then reunite with the original chromosome (Stadler[3]). Very few of the acentric fragments in Tradescantia, or in other plants, are included in the daughter nuclei, although Carlson[4] finds that acentric fragments are usually included in the daughter nuclei in the grasshopper neuroblast. The x-ray breaks induced in the chromosomes of Tradescantia either reunite in the original position, or unions occur between broken ends of different chromosomes within a maximum period of about one hour. The nuclear cycle of these cells requires about one week. Even with a nuclear cycle of several hours it seems highly improbable that acentric fragments in plants can persist and reunite with the original chromosome after several cell generations.

The exact nature of x-ray action on malignant tumors is not known, although it seems probable that nuclear injury plays some part in checking growth. Induced mutations are caused by the direct action of x-rays and not by a delayed effect. Moderate doses of x-rays have a temporary effect on the physiological processes in plant cells, but the permanent effects are caused by chromosome aberrations. The effect of the time-intensity factor on x-rayed organisms is readily explained on the basis of induced chromosome aberrations. The exponential increase of chromosome aberrations with increased dosage can be explained only as the direct effect of the radiation on the chromosomes. The greater susceptibility of prophase stages in the nuclear cycle can be attributed to purely mechanical factors. The dual nature of the chromosomes permits single hits to produce aberrations. The greater susceptibility of meiotic prophase, as compared with mitotic prophase, also can be attributed to the condition of the chromosomes. Four chromatids are closely associated so that 1-hit aberrations are more easily induced.

If we may assume that chromosome aberrations play some part in the regression of tumors following irradiation, the results of the x-ray work with plant cells may have some application to the x-ray treatment of cancer. Radiation of sufficiently low intensity would produce only 1-hit breaks, which are confined almost entirely to cells rayed at the prophase stage of the nuclear cycle. During a given interval many more prophase stages would occur in the rapidly dividing tumor cells than in the normal tissue so that most of the chromatid aberrations would be confined to the tumor cells. High concentrations of x-rays produce more aberrations at both the prophase and resting stage, but due to the fact that such intensity will produce many chromosome aberrations in resting cells, the normal tissue would be injured. Intermittent radiation of low intensity should produce frequent aberrations in the rapidly dividing tumor cells, with but few aberrations in the chromosomes of the normal tissue.

Summary.—X-ray radiation of high intensity is more effective in producing chromosome aberrations in Tradescantia microspores than the

same dosage given at low intensity. Continuous radiation is more effective than intermittent radiation. The effect of the time-intensity factor is due to the fact that the chromosome aberrations are dependent upon two independent x-ray hits, limited in time and space. Intense radiations produce many adjacent breaks during the critical time period and fusions may occur between different chromosomes. If the radiation is given slowly, the first break may heal before a second break occurs in an adjacent chromosome. The broken ends of both chromosomes reunite in the original position and no aberration is produced. The time-intensity factor has no effect on 1-hit chromatid aberrations. The broken ends of Tradescantia chromosomes may remain in an unstable condition and capable of fusion for a maximum period of about an hour.

The work was supported, in part, by a grant from the NATIONAL RESEARCH COUNCIL Committee on Radiation.

[1] Sax, Karl, *Genetics*, **23**, 494–516 (1938).
[2] Pack, Geo. T., and Edith H. Quimby, *Amer. Jour. Roentgenology*, **28**, 650–667 (1932).
[3] Stadler, L. J., *Proc. Sixth Internat. Congress Genetics*, **1**, 274–294 (1932).
[4] Carlson, J. G., *Proc. Nat. Acad. Sci.*, **24**, 500–507 (1938).

INDUCTION OF TRIPLOIDY AND HAPLOIDY IN THE NEWT, TRITURUS VIRIDESCENS, BY COLD TREATMENT OF UNSEGMENTED EGGS

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Communicated April 11, 1939

It has been known for some time that triploid embryos of frogs and salamanders occur occasionally in nature. Last year it was demonstrated that these exceptional triploid individuals may be identified among living larvae by means of chromosome counts in amputated tail-tips.[1] The evidence that has accumulated so far indicates that the frequency of triploidy varies with different species. In the newt, *Triturus viridescens*, it appears to be about one per cent (five triploids among 428 larvae). In the two-lined salamander, *Eurycea bislineata*, it may be as high as 10 per cent (13 triploids among 134 larvae examined so far).[2] Furthermore, within one species, some matings appear to produce more triploid embryos than others, a condition which suggests the participation of genetic factors.

Since the study of the effects of polyploidy in a vertebrate is a new field and has already produced interesting results,[3,4] it would be highly desirable to increase the natural frequency by experimental means. In plants, poly-

ploidy may be induced by exposure to either low or high temperatures, or by an alternating cold and heat treatment.[5-8] Under the influence of the abnormal temperatures, the anaphase separation of the chromosomes and cell division are inhibited in a greater or smaller number of cells, while the division of the chromosomes is not affected. When the treatment is applied during meiosis, diploid gametes are produced which may, by combining with normal, haploid gametes, give rise to triploid embryos. Tetraploid seedlings are obtained by application of extreme temperatures at the time of the early divisions of the zygote.

Recent investigations indicate that, in animals also, polyploidy may be induced by abnormal temperatures. Tetraploid silkworms have been obtained by short heat treatments.[9] In parthenogenetic races of *Artemia salina*, a prolonged exposure of eggs during the single maturation division to temperatures of +4°C. increases the natural tendency of some eggs to retain the polar body.[10]

The experiment to be reported here was suggested by the work of Rostand on the influence of low temperatures on crosses between various species of frogs and toads.[11-13] In some of these crosses, the foreign sperm penetrates and activates the egg; the sperm chromatin, however, does not take any part in development. Cleavage takes place with the maternal chromosomes only (gynogenesis) and usually gives rise to haploid, dwarfed tadpoles. Occasionally, however, vigorous, diploid tadpoles are produced which must have developed from diploid eggs. These are probably the same exceptional eggs which, following normal fertilization by a sperm of the same species, produced the occasional triploid embryos mentioned previously.[14] By exposing cross-fertilized eggs to temperatures close to freezing, Rostand was able to increase the number of diploid gynogenetic tadpoles. The low temperature apparently interferes with the course of the second maturation division which, in all amphibian eggs, is not completed until after fertilization: a certain number of eggs retain the chromosomes that would normally be extruded in the second polar body.

During the months of November and December, 1938, eggs of the newt, *Triturus viridescens*, were obtained by means of pituitary stimulation. As in other species of newts, the eggs are deposited singly and fertilized immediately before laying, by spermatozoa that are present in the spermatheca connecting with the cloaca. Freshly laid eggs were collected and placed in a refrigerator the temperature of which varied between +0.5° and +3°C. After sixteen to twenty-six hours, they were removed and allowed to develop at room temperature (20° to 23°C.). The rate of mortality was much higher than in untreated control eggs (table 1), but varied greatly between groups of eggs obtained from different females. When the larvae had hatched and reached an age of about three weeks, the tail-tips were amputated and stained for chromosome counts (table 2). All

but one of the normal larvae obtained from refrigerated eggs were found to be triploid, while the control larvae from untreated eggs were without exception diploid. The remaining ten experimental larvae, which were characterized by small size and varying degrees of edema, were all haploid.

TABLE 1

SUMMARY OF DEVELOPMENT OF EGGS EXPOSED AFTER FERTILIZATION TO +0.5 TO +3.0°C. FOR 16 TO 26 HOURS

	REFRIGERATED EGGS	CONTROL EGGS
Total	114	146
Died before end of gastrulation	64	10
Died in early embryonic stages	15	19
Normal larvae	25	117
Dwarfed, more or less edematous larvae	10	...

TABLE 2

SUMMARY OF CHROMOSOME COUNTS IN TAIL-TIPS OF LARVAE

	FROM REFRIGERATED EGGS	FROM CONTROL EGGS
Total	35	117
Diploid	1	117
Triploid	24	...
Haploid	10	...

Figure 1 shows typical metaphase plates from a haploid, a diploid and a triploid tail-tip, with eleven, twenty-two and thirty-three chromosomes, respectively. The size of the nuclei and cells in the epidermis of the tail-tips is roughly proportional to the chromosome number (figure 2). This is clearly shown in living larvae by the size and number of pigment cells in corresponding areas of the head (figure 3). In the haploid larva, the melanophores are much smaller but more numerous than in the diploid. In the triploid, the increase in cell size and reduction in cell number is most clearly shown in the region between the gills.

The general appearance of the same three larvae is illustrated in figure 4. The haploid larva is shorter; the greater width is largely caused by the edematous swelling of the body. The triploid larva is slightly larger than the control. This increase in body size, however, is not typical for triploid larvae in general, since many were not noticeably larger than diploid larvae of the same stage of development. In the triploid larvae obtained from untreated eggs of both *Triturus* and *Eurycea* which were mentioned above, triploidy is usually not connected with gigantism.[1,2]

The majority of the triploids developed normally to metamorphosis. Eight of the ten haploid larvae died during the fourth week. One only lived for 114 days. The presence of groups of larger, presumably diploid nuclei in five of the haploid tail-tips indicates that diploid sectors will be found in other regions of the body as well; some of these larvae are therefore not pure haploids.

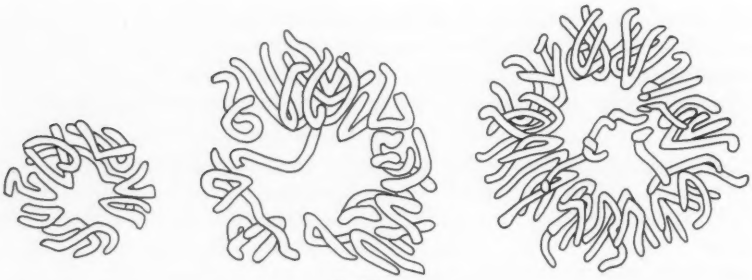

FIGURE 1

Metaphase chromosomes from epidermal cells of a haploid, a diploid and a triploid tail-tip (11, 22 and 33 chromosomes). Tracings of enlarged photomicrographs. × 1255.

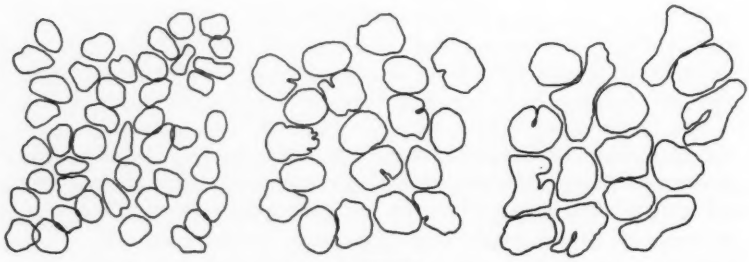

FIGURE 2

Nuclei of epidermis cells from a haploid, a diploid and a triploid tail-tip. The size of the nuclei is roughly proportional to the number of chromosomes they contain. Camera lucida drawings. × 274.

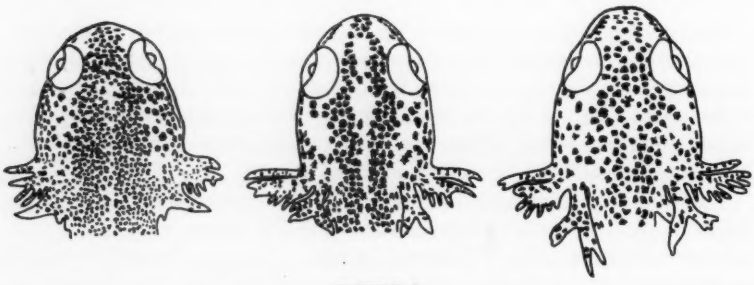

FIGURE 3

Pigment pattern on the head of a haploid, a diploid and a triploid larva, 4 weeks old. Tracings of enlarged photomicrographs. × 11.5.

The results of the refrigeration experiments that have been carried out so far demonstrate that the production of triploid salamander larvae may be controlled effectively by this simple procedure. At the same time they unexpectedly reveal a new method for inducing haploidy in animals which may have important applications. It is interesting to note that recently a haploid rye plant has been obtained by short exposures to a temperature of $-3°C.$[15]

We hope that the continuation of these experiments will determine more accurately the conditions which lead either to addition or to subtraction of a chromosome set in the treated eggs. Variations in the duration of the

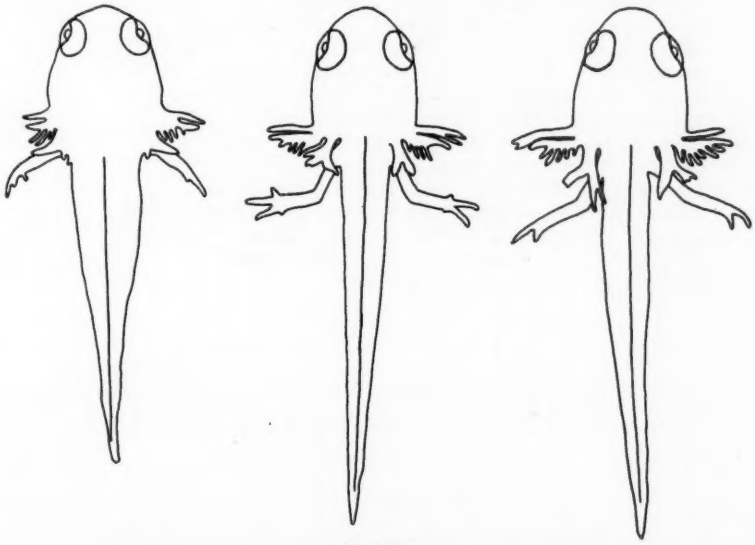

FIGURE 4

The same larvae as shown in figure 3. The haploid larva (left) is dwarfed and edematous, the triploid is slightly larger than the diploid. Tracings of enlarged photomicrographs. × 8.7.

treatment, in the temperature that is used and in the stage of fertilization when it is applied to the eggs, may yield important information. A detailed cytological study will also be made of the changes that are induced by the low temperature in the processes of maturation and fertilization. The cytological situation may be expected to be rather complex since fertilization in salamanders is normally polyspermic and the low temperature may influence the reactions of the supernumerary spermatozoa.

Before such an investigation is made it is not possible to offer a definite explanation of the mechanisms that are involved in producing triploidy or

haploidy. It is probable that the former is due to the retention of the haploid set of chromosomes that is normally eliminated in the second polar body. Under slightly different conditions, possibly when the temperature is closer to 0°C., the increased viscosity of the cytoplasm might inhibit the movements and subsequent fusion of the pronuclei. The sperm nucleus, which is normally associated with a division center, would then divide alone, and development would proceed with the haploid, paternal set of chromosomes.

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THE RÔLE OF DEVELOPMENTAL RATES IN THE PRODUCTION OF NOTCHED WING CHARACTERS IN DROSOPHILA MELANOGASTER

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Communicated March 20, 1939

In two recent papers Beadle,[1] Ephrussi[2] and co-workers described the effect of starvation, peptone-food and media with low food-levels during the larval stage on the pigmentation of the eyes of adult flies in *Drosophila melanogaster*. If the larvae developed under these abnormal conditions a change in genetical *v*-flies to normal eye-color was observed. The authors considered that a change in the metabolism of these flies was responsible for the production of v^+-substance, which is necessary to change *v*-eyes to normal eyes. No similar change could be produced in *cn*-flies.

Using the technique described by Beadle[1] and Ephrussi,[2] we tested the influence of starvation and different food-media during the larval stage on the development of different notched wing-mutants of *D. melanogaster*.

FIGURE 1a–g

Degrees of notch-effect [(0)–(5)] of cd^n flies hatching on successive days (10–17 days after egg-laying). Time of development prolonged by transfer of larvae to peptone-food 24–48 hours after egg-laying.

The investigations are still in an early stage, but the preliminary results are so clear that a short account appears justified.

Stocks of vg^{no} and ct^{n} (Fig. 1a), two low alleles of *vg* and *ct*, respectively, which have been inbred for many years and always show only a slight notch at the tip of the wing, were used in these experiments. The parental flies were allowed to lay eggs for 24 hours on a standard agar-molasses-cornmeal-yeast medium, from which the young larvae were collected at various ages and transferred to the different media. The investigation was started with two sets of experiments: (1) transfer of larvae to petri-dishes which contained wet filter-paper + 3 drops of a yeast-solution ("starvation") with return of the larvae to normal food after 3 days, or, in one test, no return at all, and (2) transfer of larvae to vials with a medium consisting of a 10% peptone-solution, glucose and agar-agar as described by Ephrussi[2] and no return to normal food. In all these experiments a significant increase of from 1–6 days in the time of development could be observed. The flies which hatched after a development thus prolonged showed a stronger notch-effect, which increased with the increase in the time of development, producing a whole series of wing-patterns, ranging from slight notches to deeply-cut wings. Examples are shown in figure 1 and are cited in table 1. The notch-effect observed in flies with a large increase in time of development, especially that produced by peptone-food, is as extreme as the effect of higher alleles of the *vg*- or *ct*- series. Current tests with larval food of different yeast-levels with or without glucose show the same effect, namely, the time of development is prolonged and the notch-effect is enhanced accordingly. This suggests that the degree of notching is dependent on the time of development, which can be prolonged by starvation and larval food-media deficient in some as yet unknown components. A similar relation between time of development and abnormal wing-structure has also been detected by genetical means by Mr. E. Gardner working in this laboratory on the effect of different mutations on the degree of notch-action, as yet unpublished and cited by his permission. He examined especially the action of *vg*-dominigenes,[3] which shift the dominance of the usually recessive *vg*, thus producing a notch-effect in flies heterozygous for *vg*. In these dominigene-stocks he detected a significant delay in the time of development. He recently raised heterozygous *vg*-larvae, not containing dominigenes, on peptone-food and observed notched wings in the heterozygous *vg*-flies, which hatched after delayed development. This suggests that the action of the dominigenes might be only that of prolonging the time of development, thus producing a notch-effect in a heterozygous *vg*-fly.

A more detailed and extended analysis of the whole problem is under way, including especially experiments with different mutations, different food-media and much more precisely timed tests.

TABLE 1

Increase in degree of notch-effect with prolonged development resulting from starvation and peptone-food. Age of larvae and flies in days after egg-laying (at 25°C.). Classes (0)–(5) indicate degrees of notch-effect: (0) = slight-notched = vg^{no} or ct^{n} under normal conditions, (1) = notch-effect slightly stronger, (2) = notch-effect stronger, (3) = very strong notch-effect, (4) = extreme notch-effect, (5) = most extreme notch-effect, *ct*-like.

TYPE OF EXPERIMENT	STOCK	AGE OF LARVAE AT TIME OF TRANSFER FROM NORM. FOOD	AGE OF LARVAE AT TIME OF RETURN FROM NORM. FOOD	AGE OF FLIES AT HATCHING, NUMBER OF FLIES AND DEGREES OF NOTCH-EFFECT							
				9–10	10–11	11–12	12–13	13–14	14–15	15–16	16–17
Peptone	ct^{n}	1–2	No return	1 ♂ (0)	4 ♀ 3 ♂ (0)	3 ♀ (0)	3 ♀ 2 ♂ (1–2)	2 ♀ 1 ♂ (2)	3 ♂ (3)	1 ♂ (4)	1 ♀ 1 ♂ (4–5)
Peptone	vg^{no}	2–3	No return	... ...	3 ♂ (0)	2 ♀ 3 ♂ (0)	2 ♀ 2 ♂ (0)	1 ♀ 1 ♂ (1–2)			
				9–11	10–12	11–13	12–14	13–15	14–16		
Peptone	vg^{no}	1–3	No return	2 ♀ 1 ♂ (0)	4 ♀ 4 ♂ (0–1)	2 ♀ 3 ♂ (1–2–3)	4 ♀ (3–4)				
Starvation	vg^{no}	1–3	4–6	1 ♀ (0)	...	2 ♀ 2 ♂ (0)	1 ♀ 3 ♂ (0–1)	1 ♀ (2)			
Starvation	vg^{no}	2–4	5–7				1 ♀ (0)	3 ♀ 1 ♂ (1–2)	1 ♀ 2 ♂ (2)		
Starvation	vg^{no}	2–4	5–7						2 ♀ 2 ♂ (1–2)		
Starvation	vg^{no}	2–4	No return			3 ♀ (1–2)					
Starvation	ct^{n}	1–3	4–6	1 ♂ (0)	3 ♀ 1 ♂ (0–1)	3 ♀ 2 ♂ (0–1–2)	1 ♂ (3)				

For a tentative explanation of our results, it is necessary to recall Goldschmidt's investigations on the embryology of notched wings.[4] He has shown that wings, which exhibit a low degree of notching will first develop normally in the very young pupae and that then a destructive process sets in which destroys the later notched parts of the wing. The larger the degree of notched, the earlier this process of destruction will start and, in cases of strong degrees of notched, the process will take place in the late larval stages. He assumed that this destructive process might be caused by a destructive substance entering the at first normally developing wing. Our experiments have shown that we can produce a series of various degrees of destruction by changing the time of development. Assuming the existence of a destructive substance, it can be inferred that by prolonging the time of development either a destructive factor is given more time to work on the developing wing or more time for the production of a destructive factor is provided. This would indicate that for the production of most notch or cut wing-patterns only two main factors may be responsible, namely, the presence of a destructive factor and the rate of development. The coördination and the different intensity of these two factors and the different stages in development at which they can act may produce the many different notch characters known in Drosophila. Furthermore, as soon as a destructive factor is present, i.e., in a heterozygous *vg*-individual, notched wings can be produced, dependent on the rate of development. This would explain the production of notched wings in heterozygous *vg*-individuals by dominigene-action or by delay in development due to peptone-food.

Whether or not there is a possible relation between our results and those of Beadle[1] and Ephrussi[2] cannot be decided at this time.

* Assistance rendered by the personnel of WPA Official Project 465-03-3-192 is acknowledged.

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INFLUENCES OF LIMITING MAGNITUDE UPON METEOR FREQUENCY

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These comments are intended to demonstrate the importance of considering the limiting naked-eye magnitude in a statistical discussion of meteor frequencies. The total number of meteors, **N**, that an observer will record, is

$$\mathbf{N} = n_l + n_{l-1} + n_{l-2} + \ldots \tag{1}$$

where n_l is the observed frequency in the magnitude interval above the limiting stellar magnitude. Observational effects reduce the observed frequency in a given magnitude interval to some fraction, f, of the true frequency, N:

$$n_l = f_l N_l. \tag{2}$$

Then equation (1) becomes

$$\mathbf{N} = f_l N_l + f_{l-1} N_{l-1} + f_{l-2} N_{l-2} + \ldots \tag{3}$$

Preliminary studies[1] indicate that for homogeneous data the relationship

$$N_a = \chi N_{a-1} = \chi^2 N_{a-2}, \text{ etc.}, \tag{4}$$

or in general

$$N_a = \chi^{\Delta m} N_{a-\Delta m}, \tag{4'}$$

holds over a wide magnitude range. With equation (4) we may rewrite (3) as

$$\mathbf{N}_a = N_a(f_l + \chi^{-1} f_{l-1} + \chi^{-2} f_{l-2} + \ldots) \tag{5}$$

or

$$\mathbf{N}_a = N_a F \tag{5'}$$

where F remains constant as the limiting magnitude changes. The total number observed with some other limiting magnitude, $a - \Delta m$, will be

$$\mathbf{N}_{a-\Delta m} = N_{a-\Delta m} F. \tag{6}$$

Then dividing (5') by (6) and substituting from (4'), we find

$$\frac{\mathbf{N}_a}{\mathbf{N}_{a-\Delta m}} = \chi^{\Delta m}. \tag{7}$$

Numerically the frequency characteristic, χ, depends upon the magnitude scale characteristic of the observer and apparently also upon the dispersion

in the velocities of the meteors. For meteor showers, in which all the particles have the same velocity, χ is usually between 2.5 and 2.7, while for sporadic meteors χ tends to be greater. From (7) we conclude that the intensities of a meteor shower as observed at two places reduced to identical conditions except for one unit in the limiting magnitudes will differ by a factor of approximately 2.5. Obviously any attempt to determine the "absolute" intensity of a shower requires a precise knowledge ($\pm 0^m.1$) of the limiting magnitude as seen by each observer. Unfortunately even the most extensive reports frequently lack any mention of the limiting magnitude.

In the reduction of observations the frequency characteristic, χ, may be separated from the observational factors, f, in several ways. The values of f measure the fraction of the observing field in which meteors of a given magnitude above the limiting magnitude are seen. For meteors four or more magnitudes brighter than the limit an average observer seems able to cover a field 60° in diameter, but fainter meteors are seen only near the center of the field. Thus the value of χ may be determined from the relative frequencies of the brighter meteors. By limiting the field of observation (to perhaps 40° diameter) the limiting magnitude to which the census will be complete will become fainter, and χ may again be determined directly from the observed frequencies. With a limited field whose diameter is approximated by the lengths of the meteor trails, which vary with the magnitude, corrections for the number of meteors moving into the field will be necessary.[2]

By Öpik's "Double Count Method"[3] the observational factors may be evaluated and the true frequencies determined for the entire magnitude range observed. Simultaneous naked-eye and telescopic observations may also be employed to indicate the observational factors for the naked-eye observer.[2]

Many problems in meteoric astronomy are being investigated on the basis of diurnal and annual variations in the meteoric intensity. Equation (7) indicates the extent to which changes in the limiting magnitude, at a given location and between individual observers, will influence the apparent frequency. Until the observations used in these investigations are reduced to a standard magnitude limit, the significance of the conclusions derived from them must be impaired.

Since certain investigators describe their magnitude observations by the mean magnitude, we need to evaluate the change in mean magnitude that results from a change in the limiting magnitude. In terms of (5) and (5′) the average magnitude $\mathbf{M}$, for limiting magnitude a, is

$$\mathbf{M}_a = \frac{af_i + (a-1)f_{i-1}\,\chi^{-1} + (a-2)f_{i-2}\,\chi^{-2} + \cdots}{F} = \frac{H}{F}. \quad (8)$$

For limiting magnitude $a - \Delta m$ the mean magnitude is

$$\mathbf{M}_{a-\Delta m} = \frac{(a - \Delta m)f_1 + (a - \Delta m - 1)f_{1-1}\chi^{-1} + \ldots .}{F} = \frac{H - \Delta m \cdot F}{F}. \quad (9)$$

From (8) and (9) we have

$$\mathbf{M}_a - \mathbf{M}_{a-\Delta m} = \Delta m. \quad (10)$$

Thus the change in the mean magnitude equals the change in the limiting magnitude and is independent of both χ and the observational factors. If χ is only slightly greater than unity, the convergence in the series denoted as F and H will be slow, but in all data investigated thus far χ exceeds two. Thus in practice the series F and H converge rapidly.

[1] *H. B.* 895 (1934); *H. Ann.*, **105**, *Tercentenary Paper* 32 (1937); and unpublished
[2] Watson, *Proc. Am. Phil. Soc.*, in press.
[3] *Pub. Tartu Obs.*, **25**, no. 1 (1922); no. 4 (1923).

IS AUXIN PRODUCED IN ROOTS?[1]

BY J. VAN OVERBEEK

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Communicated March 27, 1939

In 1933 Boysen Jensen[2] conclusively demonstrated that auxin is present in roots. When this problem was solved a new one arose: Is auxin produced in root tips, or is it merely accumulated there from elsewhere in the plant? Thimann[3] determined the amount of auxin which was given off by excised tips of Avena roots into dextrose-agar blocks and compared it with the amount of auxin that was obtained by ether extraction of such tips. In both cases he found the same amount of auxin and concluded that it is merely accumulated in the Avena roots and not actually produced by them. Other authors,[4] however, found that from the roots of Avena and other plants two to twenty times as much auxin could be obtained by diffusion as by extraction. They came to the conclusion that auxin is produced in roots. As we had obtained surprisingly large amounts of auxin from pea roots[5] it seemed of interest to compare the amounts of auxin obtained from excised root tips of germinating peas by diffusion and by extraction with improved techniques.

Tips 3 to 4 mm. long were cut from roots of "Alaska" peas which had germinated for two days in washed sand. These tips were placed on agar blocks ($1^1/_2$ per cent) containing 10 per cent dextrose, and were replaced each hour on fresh blocks. After from 6 to 9 hours no more auxin was given

off into the blocks (Fig. 1), not even if the tips were left on a single block for an additional period of ten hours. Yet at the end of such a prolonged diffusion experiment the tips still contained some auxin which could be demonstrated by ether extraction for 20 hours in a Soxhlet extractor (table). A similar set of roots as was used for the diffusion test was extracted with highly purified ether in a small Soxhlet extractor for 20 hours immediately after they were cut (Fig. 2). The amount of auxin obtained

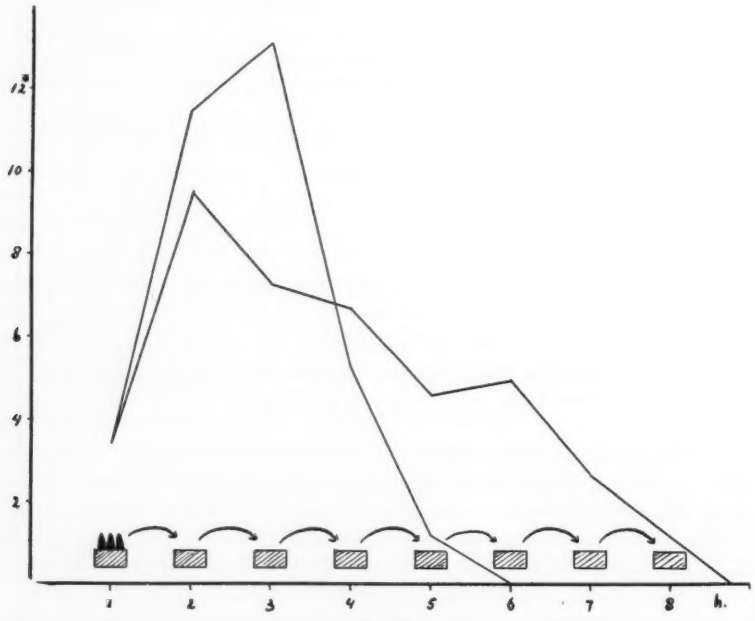

FIGURE 1

Amount of auxin (degrees of curvature in the Avena test) given off by 20 tips of pea roots during periods of 1 hour in 140 mm.³ agar ($1^1/_2\%$) blocks containing 10% dextrose. (80923–28.)

in this way was practically equal to the sum of the amounts obtained in the diffusion test followed by extraction. It is interesting to note that diffusion yields less auxin than direct extraction, contrary to the results obtained by all previous investigators. It therefore seems that, under the conditions of our experiments, the auxin given off by *excised root tips of germinating peas* is auxin which was already present at the time the tip was cut off, and is not produced. Of further interest is the fact that in these root tips there is some auxin which apparently is not able to diffuse

out, but can be demonstrated by extraction only, which lends support to the view expressed by Thimann and Went[6] that auxin in the plants occurs in a bound and a free form.

TABLE

Total amount[7] of auxin obtained by:	
Diffusion from 50 tips	66.4° ± 2.6° (= 69% free moving auxin)
Ether extraction of residual auxin from same tips	29.6° ± 0.3° (= 31% bound auxin)
Total	96.0°
Direct ether extraction from 50 tips	91.4° ± 2.6°

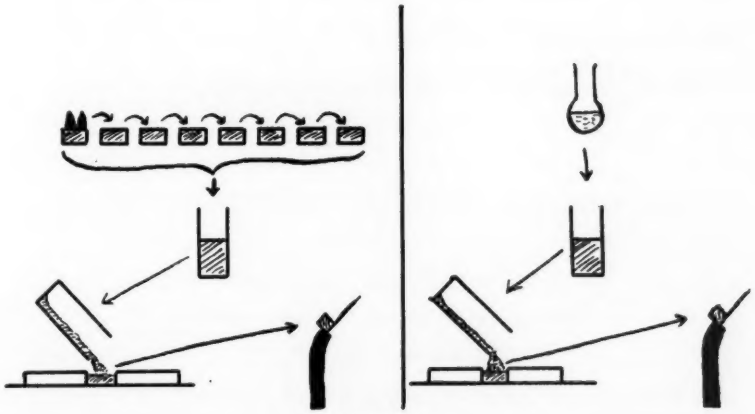

FIGURE 2

Scheme of the technique followed to obtain auxin from root tips of germinating peas. On the left the diffusion method consisting of: (1) the collection of auxin in dextrose-agar blocks, (2) melting of these blocks, in order to make the results obtained by this method strictly comparable to those obtained by the extraction method, (3) pouring of the agar into standard size blocks, (4) determination of the auxin content by means of the Avena test. On the right the extraction method which consists of: (1) the Soxhlet extraction, (2) the transfer of the extract into hot dextrose-agar (dextrose is used in order to make the extraction technique as comparable as possible to the diffusion method), (3) pouring of blocks of standard size, (4) the Avena test.

Although under the conditions described above (excised roots on dextrose-agar blocks), no evidence was found for auxin production, this does not necessarily mean that under natural conditions (intact roots) no production will take place. Recently there has appeared to be some evidence that excised pea roots grown *in vitro* under sterile conditions and in a

complete medium[8] (containing both vitamin B_1 and nicotinic acid) are able to synthesize auxin. Details will be published later.

[1] Report on work carried out with the aid of assistants supplied by the Works Progress Administration, Official Project Number 665-07-3-83, Work Project L-9809.

[2] *Planta*, **19**, 354 (1933).

[3] *Jour. Gen. Physiol.*, **18**, 23 (1934).

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[6] Went, F. W., and Thimann, K. V., *Phytohormones*, Macmillan, New York, 1937.

[7] The amounts of auxin are expressed in degrees of curvature in the standard Avena test on the basis of 50 root tips and a volume of agar corresponding to 8 blocks (1120 mm.[3]). All tests were done in quadruplicate and at the same time in order to avoid errors due to variation in sensitivity of the plants. If root tips are placed on plain agar blocks no auxin is found in them. Ether extraction of such tips that had been transferred to fresh plain agar blocks for a total period of 20 hours, yielded $46.6° \pm 1.6°$.

[8] Addicott, F. T., and Bonner, J., *Science*, **88**, 577–578 (1938).

CELL POLARITY AND THE DIFFERENTIATION OF ROOT HAIRS

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Communicated March 24, 1939

By means of a technique recently described by Sinnott[1] it is possible to observe the division and enlargement of cells in the living root tips of certain grasses, and to trace the history of these cells until differentiation is complete. In certain genera the cells which are to form root hairs (the trichoblasts) are sharply differentiated at the last cell division from those which are not, and the two develop very differently. In other genera differentiation of root hair cells is delayed for some time, and any cell, until rather late in its development, seems to have the capacity to form a root hair. A detailed comparison of these two types of differentiation throws light on the mechanism of root-hair determination. Root hair development was studied in *Phleum pratense*, *Poa trivialis*, *Chloris gayana* and *Sporobolus cryptandrus*.

In the genera *Phleum* and *Poa*, just before the last cell division (usually the fourth from the tip) in the surface layer or dermatogen, the apical end of the cell becomes more densely protoplasmic than the basal. The division which then follows is an unequal one, the apical (distal) daughter cell, which is the trichoblast and destined to produce a root hair, being

considerably smaller than the basal (proximal) one (Fig. 1, above). It also has denser contents, as may be seen in the living condition or by staining with Ruthenium Red or other agents. The new wall between the two cells is somewhat convex toward the proximal side, so that the cells differ more in the length of their side walls than in volume. The absolute and relative sizes of these apical and basal cells, measured along the side walls at the beginning of development and at maturity, are shown in table 1. *Phleum* has slightly larger cells, but the relative size of the two cell types is much the same as in *Poa*.

TABLE 1

	Phleum	*Poa*	*Chloris*	*Sporobolus*
MERISTEMATIC REGION				
Length of apical cell (micra)	4.68 ± 0.25	3.59 ± 0.20	6.60 ± 0.61	7.17 ± 0.21
Length of basal cell (micra)	10.33 ± 0.39	8.12 ± 0.30	9.00 ± 0.75	8.25 ± 0.26
Ratio, apical:basal	1.0 : 2.21	1.0 : 2.26	1.0 : 1.36	1.0 : 1.15
MATURE REGION				
Length of apical cell (micra)	98.3 ± 6.1	52.6 ± 4.4	95.0 ± 5.3	101.0 ± 5.2
Length of basal cell (micra)	244.8 ± 11.3	178.5 ± 8.5	137.0 ± 6.8	127.0 ± 6.8
Ratio, apical:basal	1.0: 2.49	1.0 : 3.39	1.0 : 1.44	1.0 : 1.26
Percentage distance of root hair origin from apical end of cell.				
Per cent	14.0 ± 0.6	18.0 ± 0.5	23.9 ± 0.6	41.9 ± 1.1
Standard deviation	5.83 ± 0.40	5.25 ± 0.37	5.96 ± 0.43	9.84 ± 0.76

As cell enlargement proceeds, these relative proportions are essentially maintained, although from the first the apical cell grows somewhat less rapidly than the basal so that the contrast between them increases until maturity. The partition wall loses its convex form and becomes straight. The basal cell soon becomes vacuolate but the apical one remains rather densely protoplasmic. As this cell reaches its final size, a root hair begins to develop from near the apical end, where more protoplasmic material is present. The nucleus usually occupies a position just below the young root hair. The point of origin of the hair, in terms of percentage distance along the cell from its apical to its basal end, is presented in table 1. This point, about 14% and 18% from the apical end, in these two genera, is relatively constant, showing a standard deviation of about 5.5%. The root hair itself is pointed forward at an angle of about 45° with the axis of the root.

In the genus *Sporobolus* (Fig. 1, below), on the other hand, there is no visible internal differentiation before the last division and the division itself is much more nearly an equal one, the two daughter cells usually being indistinguishable in size or contents, although there is a tendency for the apical one to be slightly smaller. As the cells expand to their final size, root hairs develop from some of them. Occasionally one finds alternating root-hair cells and hairless cells, as in the figure, but frequently there is a

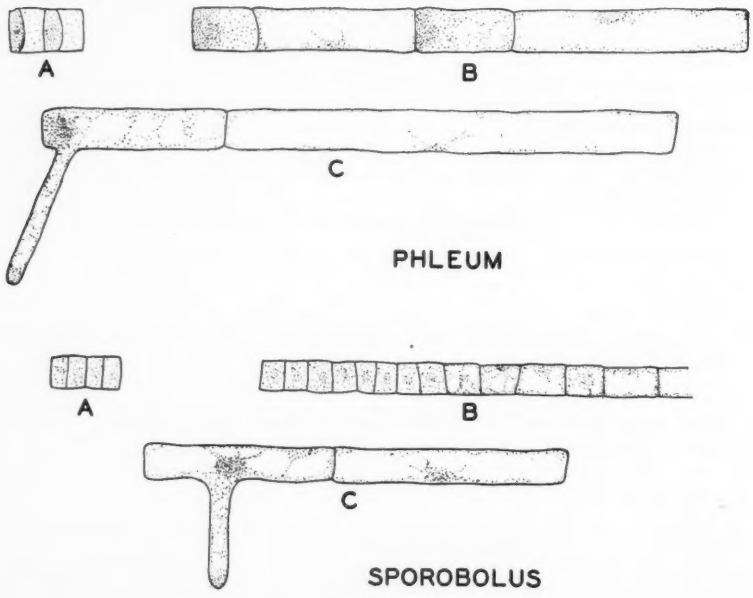

FIGURE 1

Above, *Phleum: A*, two pairs of cells in the meristematic region; *B*, the same cells during expansion; *C*, a root-hair cell and a hairless cell at maturity.

Below, *Sporobolus: A*, two pairs of cells in meristematic region; *B*, a series of cells during expansion; *C*, a root-hair cell and a hairless cell at maturity.

The apex of the root is toward the left.

continuous series of cells which develop hairs and then a group of several without them. The root hair originates near the middle of the cell, about 42% of the distance from the apical end (table 1). Its position is much more variable than in *Phleum* and *Poa*, however, the standard deviation being almost twice as great. The hair grows nearly straight out from the root at an angle of 90°.

The genus *Chloris* is somewhat intermediate between the two types just described. The last division appears to be slightly more unequal, the

apical cell being smaller than the basal, but this difference is by no means as great as in *Phleum* and *Poa*. As the cells enlarge, certain of them can be shown by appropriate staining to have more dense contents, and these are usually the smaller cells. They often alternate with somewhat larger ones, and are the cells from which root hairs develop. Frequently there may be a series of several successive root-hair cells. The hairs come out from about a fourth (23.9%) of the way back along the cell and are relatively constant in their position. They tend to bend forward apically somewhat.

In both *Sporobolus* and *Chloris*, roots which have been checked in their growth, because of age or for other reasons, tend to show a much more definite alternation of relatively small root-hair cells with relatively large hairless ones, and the hairs in such cases tend to originate nearer the apex of the cell than they do in fast-growing roots.

A comparison of these types of root-hair differentiation shows that early differentiation is associated with (1) denser cell contents at the apical end of the mother cell before its division into root-hair and hairless cells; (2) unequal division of this cell, the smaller daughter cell (which will produce the root hair) lying toward the apex and retaining denser cytoplasmic contents for a considerable time during subsequent extension; (3) an origin of the root hair near the apical end of the cell and in a relatively constant position; and (4) a forward growth of the root hair at an acute angle with the root axis. Conversely, where differentiation is late, the cell contents of the mother cell are of equal density throughout; the division of this cell is essentially equal so that there is less regular alternation of root-hair and hairless cells; the hair originates from near the middle of the cell but in a much more variable position, and the hair tends to grow directly out at right angles to the root axis.

These results can be explained by assuming that in *Phleum* and similar forms there is a strong apical tendency in the formation of root hairs, but very much weaker tendency in *Sporobolus* and its allies. This can perhaps best be pictured in terms of the distribution of materials under the influence of cell polarity. Where the cell is strongly polarized, the materials at one end will be markedly different from those at the other. Where polarization is weaker, distribution will be more nearly equal. If materials which stimulate root-hair formation move under the influence of strong polarity, they will accumulate at one end of the cell (the apical end in *Phleum* and its allies) from a very early stage and will cause the last division of the root hair mother cell to be unequal, the apical cell to be the root-hair cell, and the root hair itself to originate near the apical end of this cell in a constant and apically directed position. Conversely, the more equal distribution of these materials would tend to be associated with a division of the mother cell into cells essentially equal in size and in root-hair potency, and in the origin of an outward pointing root hair near the middle of the cell but more

variable in its position, exactly the situation which occurs in *Sporobolus*. In such cases of weak polarization it is probably differences in the internal or external environment rather than inherent differences in the cells themselves which determine whether a given cell will produce a root hair or not, and where it will be produced.

The material stimulating root-hair growth may be protoplasm itself or some more specific growth substance. In support of the former possibility is the fact that in *Phleum* and *Poa*, protoplasm and nucleus tend to move toward the apical end of the mother cell and of the root hair cell itself.

Aside from its effect on the distribution of stimulating materials, cell polarity might have an influence on root-hair production through differences in permeability at the two ends of the cell, through polar differences in the character of the cell wall, or through other factors affecting the form and development of the cell itself.

These pronounced and regular differences between cells in their capacity to form root hairs, and between related genera in the time and character of root-hair differentiation, should be taken into account in studies which deal with the factors responsible for root-hair development.

Whatever the causes may be for the facts here described, it is clear that in related genera of the grass family there are markedly different types of root-hair determination. In one, differentiation is early and strongly apical in character. In another it is late and but slightly or not at all apical. These forms evidently provide excellent material for experimental studies on some of the factors involved in differentiation, for they present a simple example of two sister cells which in some cases have the same fate throughout, in others become unlike only near the end of their growth, and in still others are differentiated, both in structure and function, very early in development.

[1] Sinnott, Edmund W., *Proc. Nat. Acad. Sci.*, **25**, 55–58 (1939).

ON MODULAR AND $\mathfrak{p}$-ADIC REPRESENTATIONS OF ALGEBRAS

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1. In the usual treatment of the arithmetic of an algebra A, only maximal domains of integrity are considered. However, in the important case of the group ring belonging to a group G of finite order, a domain of integrity J which is not a maximal domain is defined in a natural manner. A basis of J is formed by the group elements. It is necessary for the in-

vestigation of the modular representations of G to study the arithmetic of J instead of first replacing J by a maximal domain. We consider ground fields K which are algebraic number fields (though almost all the results could easily be extended to fields K with a discrete valuation). Let $\mathfrak{p}$ be a prime ideal of K. We are mainly interested in the connection between the $\mathfrak{p}$-adic behavior of an algebra A over K and the prime ideal divisors $\mathfrak{P}$ of $\mathfrak{p}$ in a domain of integrity J of A.

2. There are three different ways of expressing certain facts concerning algebras. We may use the language of the theory of representations, of algebras and of ideals. We first give some results concerning representations and indicate the connection with the two other theories.

Let A be an algebra with a principal unit 1 over any ground field K. Let $\mathfrak{U}_1, \mathfrak{U}_2, \ldots, \mathfrak{U}_k$ be the distinct (i.e., nonsimilar) indecomposable constituents of the first regular representation $\mathfrak{R}$ of A.[1] The largest completely reducible constituent $\mathfrak{F}_\kappa$ at the bottom of $\mathfrak{U}_\kappa$ is irreducible; $\mathfrak{F}_1, \mathfrak{F}_2, \ldots, \mathfrak{F}_k$ are distinct, and there are no other irreducible representations of A in the field K. Similarly, there exists exactly one indecomposable constituent $\mathfrak{V}_\kappa$ of the second regular representation $\mathfrak{S}$ of A which has $\mathfrak{F}_\kappa$ as the largest completely reducible constituent at the top. $\mathfrak{V}_1, \mathfrak{V}_2, \ldots, \mathfrak{V}_k$ are then all the distinct indecomposable constituents of $\mathfrak{S}$. We denote the degrees of $\mathfrak{F}_\kappa$, $\mathfrak{U}_\kappa$, $\mathfrak{V}_\kappa$ by f_κ, u_κ, v_κ, respectively. Let r_κ be the number of linearly independent matrices in K which commute with every element of $\mathfrak{F}_\kappa$. Then $h_\kappa = f_\kappa/r_\kappa$ is an integer, and $\mathfrak{U}_\kappa$ appears exactly h_κ times as an indecomposable constituent of $\mathfrak{R}$, $\mathfrak{V}_\kappa$ appears h_κ times as an indecomposable constituent of $\mathfrak{S}$. On the other hand, $\mathfrak{F}_\kappa$ has the multiplicity v_κ/r_κ as an irreducible constituent of $\mathfrak{R}$, and the multiplicity u_κ/r_κ as an irreducible constituent of $\mathfrak{S}$.

The multiplicity of $\mathfrak{F}_\lambda$ as a constituent of $\mathfrak{U}_\kappa$ will be denoted by $c_{\kappa\lambda}/r_\lambda$. We write

$$\mathfrak{U}_\kappa \longleftrightarrow \sum_{\lambda=1}^{k} \frac{c_{\kappa\lambda}}{r_\lambda} \mathfrak{F}_\lambda, \qquad (\kappa = 1, 2, \ldots, k) \tag{1}$$

where the sign $\longleftrightarrow$ indicates that we have the same irreducible constituents on both sides. The $c_{\kappa\lambda}$ are integers which are divisible by r_κ and r_λ. They are the *Cartan invariants* of A. Corresponding to (1), we have

$$\mathfrak{V}_\kappa \longleftrightarrow \sum_{\lambda=1}^{k} \frac{c_{\lambda\kappa}}{r_\lambda} \mathfrak{F}_\lambda. \qquad (\kappa = 1, 2, \ldots, k). \tag{2}$$

On comparing the degrees in (1) and (2), we obtain formulas which express u_κ and v_κ in terms of $c_{\kappa\lambda}$, r_κ and $h_\kappa = f_\kappa/r_\kappa$.

The radical N of A consists of those elements which are represented by 0 in every $\mathfrak{F}_\kappa$. The matrices of $\mathfrak{F}_\kappa$ form a simple algebra A_κ, and the residue

class algebra A/N is isomorphic to the direct sum of these A_κ, $\kappa = 1, 2, \ldots, k$. Each A_κ is a complete matric algebra of degree h_κ with coefficients in a division algebra Δ_κ of rank r_κ over K (we use the word "rank" to denote the number of basis elements of an algebra!).

We write 1 as the sum of orthogonal primitive idempotents[2] and select for every κ one such idempotent η_κ which (mod N) belongs to A_κ. Then the rank of $\eta_\kappa A \eta_\lambda$ is given by the Cartan invariant $c_{\kappa\lambda}$. It may also be remarked that if the Loewy series of $\mathfrak{R}$ or any other (1–1) representation of A consists of e terms, then $N^e = 0$ but $N^{e-1} \neq 0$.

The elements of A represented by 0 in $\mathfrak{F}_\kappa$ form a prime ideal $\mathfrak{P}_\kappa$ of A, and these ideals $\mathfrak{P}_1, \mathfrak{P}_2, \ldots, \mathfrak{P}_k$ are the only prime ideals of A. Of course, $A/\mathfrak{P}_\kappa \simeq A_\kappa$, and the intersection $[\mathfrak{P}_1, \mathfrak{P}_2, \ldots, \mathfrak{P}_k]$ is equal to N. It may happen that $\mathfrak{P}_\kappa^2 = \mathfrak{P}_\kappa$ for every κ.

3. We distribute the indecomposable constituents $\mathfrak{U}_\kappa$ into *blocks* in the following manner. The constituents $\mathfrak{U}_\rho$ and $\mathfrak{U}_\sigma$ belong to the same block if we can find a sequence $\mathfrak{U}_\rho, \mathfrak{U}_\mu, \ldots, \mathfrak{U}_\tau, \mathfrak{U}_\sigma$ which begins with $\mathfrak{U}_\rho$ and ends with $\mathfrak{U}_\sigma$, such that any two consecutive $\mathfrak{U}_\kappa$ have at least one irreducible constituent $\mathfrak{F}_\lambda$ in common. Suppose that we have t such blocks: $\mathfrak{B}_1, \mathfrak{B}_2, \ldots, \mathfrak{B}_t$. We then may also speak of the constituents $\mathfrak{F}_\lambda$, or the prime ideals $\mathfrak{P}_\lambda$, of $\mathfrak{B}_\tau$.

To every block $\mathfrak{B}_\tau$ there belongs (*a*) the set $\mathfrak{N}_\tau$ of those elements of A which are represented by 0 in every $\mathfrak{U}_\lambda$ outside of $\mathfrak{B}_\tau$, and (*b*) the set $\mathfrak{M}_\tau$ of those elements which are represented by 0 in all $\mathfrak{U}_\lambda$ of $\mathfrak{B}_\tau$. The sets $\mathfrak{N}_\tau$ and $\mathfrak{M}_\tau$ are ideals of A, and, of course, $A = \mathfrak{M}_\tau \oplus \mathfrak{N}_\tau$. Then

$$A = \mathfrak{N}_1 \oplus \mathfrak{N}_2 \oplus \ldots \oplus \mathfrak{N}_t,$$

where the $\mathfrak{N}_\tau$ cannot be written as direct sum. Correspondingly, the intersection $[\mathfrak{M}_1, \mathfrak{M}_2, \ldots, \mathfrak{M}_t]$ is the 0-ideal (0). The ideals $\mathfrak{M}_i$ and $\mathfrak{M}_j$ are relatively prime: $(\mathfrak{M}_i, \mathfrak{M}_j) = A$ for $i \neq j$. Finally, $\mathfrak{M}_i$ cannot be written as the intersection of two relatively prime ideals $\neq \mathfrak{M}_i$. This is the only representation of (0) as an intersection of ideals where all these properties hold.

4. We assume from now on that K is an algebraic number field. (With slight modifications, the following results can be derived for any field K with a discrete valuation.) Let J be a domain of integrity in the algebra A of rank n in the following sense: (1) J is a subring of A; (2) J contains n linearly independent elements of A; (3) the elements of J when expressed by a basis $\epsilon_1, \epsilon_2, \ldots, \epsilon_n$ of A have the form $\Sigma a_i \epsilon_i$ with $a_i = b_i/w$, where w is a fixed denominator in K and the b_i are integers of K; (4) J contains the ring $\mathfrak{o}$ of all integers of K.

Every ideal $\mathfrak{m}$ of $\mathfrak{o}$ generates an ideal of J which without danger of confusion may be denoted by $\mathfrak{m}$ again. Let $\mathfrak{p}$ be a fixed prime ideal of $\mathfrak{o}$.

We denote by $\mathfrak{o}^*$ the ring of all $\mathfrak{p}$-integers of K, i.e., of all a/b, where a, b lie in $\mathfrak{o}$ and $(b, \mathfrak{p}) = \mathfrak{o}$. Then $\mathfrak{o}^*$ and J generate a subring J^* of A. Here J^* has a basis $\eta_1, \ldots, \eta_n$ such that every γ of J^* can be written uniquely in the form

$$\gamma = c_1\eta_1 + c_2\eta_2 + \ldots + c_n\eta_n, \qquad c_i \text{ in } \mathfrak{o}^*. \tag{3}$$

The η_i can be chosen in J.

The ideal $\mathfrak{p}$ generates an ideal of $\mathfrak{o}^*$ and an ideal of J^*, both of which will be denoted by $\mathfrak{p}^*$. The element γ in (3) belongs to $\mathfrak{p}^*$, if all the c_i are of the form a_i/b_i with a_i, b_i in $\mathfrak{o}$, $a_i \equiv 0 \pmod{\mathfrak{p}}$ and $(b_i, \mathfrak{p}) = \mathfrak{o}$. We denote quite generally the residue class of an element α of $J^* \pmod{\mathfrak{p}^*}$. by $\overline{\alpha}$. We have

$$\bar{\mathfrak{o}} = \mathfrak{o}^*/\mathfrak{p}^* \cong \mathfrak{o}/\mathfrak{p}; \qquad \overline{J} = J^*/\mathfrak{p}^* \cong J/\mathfrak{p} \tag{4}$$

for the residue class field and residue class algebra. The elements $\overline{\eta}_1$, $\overline{\eta}_2, \ldots, \overline{\eta}_n$ form a basis of $\overline{J}$ with regard to $\bar{\mathfrak{o}}$. If $B = (b_{\kappa\lambda})$ is a matrix with coefficients in $\mathfrak{o}^*$, then the corresponding matrix $(b_{\kappa\lambda})$ with coefficients in $\bar{\mathfrak{o}}$ will be denoted by $\overline{B}$. The same notation will be used in the case of representations.

We form the regular representation $\mathfrak{R}$ of A, using the basis $\eta_1, \eta_2, \ldots, \eta_n$. Every γ of J^* is then represented by a matrix $R(\gamma)$, with coefficients in $\mathfrak{o}^*$. If $\gamma_1 \equiv \gamma_2 \pmod{\mathfrak{p}^*}$, then $R(\gamma_1) \equiv R(\gamma_2) \pmod{\mathfrak{p}^*}$. This implies that $\bar{\gamma} \to R(\bar{\gamma})$ gives a representation $\overline{\mathfrak{R}}$ of $\overline{J} = J^*/\mathfrak{p}^*$. Obviously, $\overline{\mathfrak{R}}$ is the regular representation of $\overline{J}$, formed by means of the basis $\overline{\eta}_1, \overline{\eta}_2, \ldots, \overline{\eta}_n$.

We use for $\overline{\mathfrak{R}}$ (instead of $\mathfrak{R}$) the notations introduced for the regular representation above. The k irreducible constituents $\mathfrak{F}_\kappa$ of $\overline{\mathfrak{R}}$ correspond to the k prime ideals $\overline{\mathfrak{P}}_\kappa$ of $\overline{J} \cong J/\mathfrak{p}$. It follows that there exist exactly k prime ideal divisors $\mathfrak{P}_\kappa$ of $\mathfrak{p}$ in J. Here $\mathfrak{P}_\kappa$ consists of those γ of J for which $\bar{\gamma}$ is represented by 0 in $\mathfrak{F}_\kappa$. The residue class algebra $J/\mathfrak{P}_\kappa$ is represented by $\mathfrak{F}_\kappa$; it is a complete matric algebra of degree h_κ in a Galois field of degree r_κ over $\bar{\mathfrak{o}} \cong \mathfrak{o}/\mathfrak{p}$. The intersection of the $\mathfrak{P}_\kappa$ gives the radical of $\mathfrak{p}$.

The t blocks $B_1, B_2, \ldots, B_t$ of $\overline{\mathfrak{R}}$ give t ideals $\overline{\mathfrak{M}}_1, \overline{\mathfrak{M}}_2, \ldots, \overline{\mathfrak{M}}_t$ of $J/\mathfrak{p}$. For the corresponding ideals of $\mathfrak{M}_\tau$ of J, we have

$$\mathfrak{p} = [\mathfrak{M}_1, \mathfrak{M}_2, \ldots, \mathfrak{M}_t]; \tag{5}$$

any two of the $\mathfrak{M}_\tau$ are relatively prime, and $\mathfrak{M}_\tau$ cannot be written as intersection of relatively prime ideals $\neq \mathfrak{M}_\tau$. (5) is the only representation of $\mathfrak{p}$ as intersection of ideals with all these properties.

5. Let π be an element of $\mathfrak{o}$ such that $\pi \equiv 0 \pmod{\mathfrak{p}}$, $\pi \not\equiv 0 \pmod{\mathfrak{p}^2}$. It follows easily from the connection between $\mathfrak{R}$ and $\overline{\mathfrak{R}}$ and the structure of $\overline{\mathfrak{R}}$ that we may assume

$$R(\alpha) = \begin{vmatrix} M_{11} & \pi M_{12} & \pi M_{13} & \dots \\ \pi M_{21} & M_{22} & \pi M_{23} & \dots \\ \pi M_{31} & \pi M_{32} & M_{33} & \dots \\ \dots & \dots & \dots & \dots \end{vmatrix} \tag{6}$$

for α in J^*, where $M_{\kappa\lambda}$ has coefficients in $\mathfrak{o}^*$, and $\overline{M}_{11}, \overline{M}_{22}, \overline{M}_{33}, \dots$ coincide with $\mathfrak{U}_1, \mathfrak{U}_2, \dots$ for the element α, each of the latter taken with the proper multiplicity h_κ. In (6) a chain of similarity transformations of determinant 1 can be applied such that we obtain corresponding formulas with π^i instead of π, $i = 2, 3, 4, \dots$. This can easily be seen by means of the properties of intertwining matrices. We now replace the ground field K by the corresponding $\mathfrak{p}$-adic field $K_\mathfrak{p}$. The ideal (π) generated by $\mathfrak{p}$ will again be denoted by $\mathfrak{p}$, a bar again indicates the transition from an element to its residue class (mod $\mathfrak{p}$). It follows then that in $K_\mathfrak{p}$, $R(\alpha)$ in (6) can be transformed in

$$\begin{vmatrix} M_1 & 0 & 0 & \dots \\ 0 & M_2 & 0 & \dots \\ 0 & 0 & M_3 & \dots \\ \dots & \dots & \dots & \dots \end{vmatrix}$$

by similarity transformations of determinant 1, where $\overline{M}_1, \overline{M}_2, \dots$ again are identical with $\mathfrak{U}_1, \mathfrak{U}_2, \dots$ for the element α, each taken with the proper multiplicity h_κ. This shows the existence of a *representation* $(\mathfrak{U}_\kappa)$ *of* A *with coefficients in the* $\mathfrak{p}$*-adic field* $K_\mathfrak{p}$, *such that we obtain the indecomposable modular representation* $\mathfrak{U}_\kappa$ *of* $J/\mathfrak{p}$ *when we replace every coefficient in* $(\mathfrak{U}_\kappa)$ *by its residue class* (*mod* $\mathfrak{p}$):

$$\overline{(\mathfrak{U}_\kappa)} = \mathfrak{U}_\kappa. \qquad (\kappa = 1, 2, \dots, k). \tag{7}$$

The regular representation $\mathfrak{R}$ of A splits completely into $(\mathfrak{U}_1), (\mathfrak{U}_2), \dots$ where $(\mathfrak{U}_\kappa)$ is to be taken h_κ-times. The $\mathfrak{p}$-adic representation $(\mathfrak{U}_\kappa)$ here may be decomposable in $K_\mathfrak{p}$. Our result can be considered as a generalization of *Hensel's irreducibility theorem*[3] for polynomials with the highest coefficient 1.

6. Let $\mathfrak{T}_1, \mathfrak{T}_2, \dots, \mathfrak{T}_l$ be the distinct irreducible representations of A in $K_\mathfrak{p}$. Using a method of *Burnside*[4] we can show that after a similarity transformation we can assume that $\mathfrak{T}_\lambda$ represents the elements of J^* by matrices with integral $\mathfrak{p}$-adic coefficients. Then $\overline{\mathfrak{T}}_\lambda$ gives a representation of $\overline{J} \cong J/\mathfrak{p}$.

Every matrix P with integral $\mathfrak{p}$-adic coefficients which intertwines $(\mathfrak{U}_\sigma)$ and $\mathfrak{T}_\rho$,

$$(\mathfrak{U}_\sigma) \cdot P = P \cdot \mathfrak{T}_\rho,$$

produces a matrix $\overline{P}$ intertwining $\overline{(\mathfrak{U}_\sigma)} = \mathfrak{U}_\sigma$ and $\overline{\mathfrak{T}}_\rho$. Using the properties

of intertwining matrices it can be shown that every matrix intertwining $\mathfrak{U}_\sigma$ and $\overline{\mathfrak{T}}_\rho$ can be obtained in this manner. For the number $I(.\ ,\ .)$ of linearly independent intertwining matrices, we have then

$$I((\mathfrak{U}_\sigma), \mathfrak{T}_\rho) = I(\mathfrak{U}_\sigma, \overline{\mathfrak{T}}_\rho), \tag{8}$$

where the left side refers to the $\mathfrak{p}$-adic field $K_\mathfrak{p}$; the right side to the modular field $\bar{\mathfrak{o}} \cong \mathfrak{o}/\mathfrak{p}$.

For a direct computation of both expressions in (8) we assume now that A is semisimple. The $\mathfrak{p}$-adic representation $(\mathfrak{U}_\sigma)$ splits completely into irreducible $\mathfrak{p}$-adic representations $\mathfrak{T}_\rho$:

$$(\mathfrak{U}_\sigma) \longleftrightarrow \sum_{\rho=1}^{l} d_{\rho\sigma}\mathfrak{T}_\rho \qquad (\sigma = 1, 2, \ldots, k) \tag{9}$$

where the $d_{\rho\sigma}$ are rational integers $\geqq 0$. On the other hand, the representations $\overline{\mathfrak{T}}_\rho$ of $\bar{J}$ can be built up from the irreducible representation $\mathfrak{F}_\kappa$ of $\bar{J}$ in the field $\bar{\mathfrak{o}}$:

$$\overline{\mathfrak{T}}_\rho \longleftrightarrow \sum_{\sigma=1}^{k} \tilde{d}_{\rho\sigma}\mathfrak{F}_\sigma \tag{10}$$

with rational integral coefficients $\tilde{d}_{\rho\sigma} \geqq 0$. We have now $q_\rho d_{\rho\sigma}$ for the left side of (8), where q_ρ denotes the number of linearly independent matrices which commute with every matrix of $\mathfrak{T}_\rho$. For the right side of (8) we find $\tilde{d}_{\rho\sigma} r_\sigma$. Hence

$$q_\rho d_{\rho\sigma} = \tilde{d}_{\rho\sigma} r_\sigma. \tag{11}$$

The indecomposable constituents $\mathfrak{U}_\sigma$, *the irreducible constituents* $\mathfrak{F}_\rho$ *of the regular representation of* $\mathfrak{J}/\mathfrak{p}$, *and the irreducible* $\mathfrak{p}$*-adic representations* $\mathfrak{T}_\lambda$ *of* A *are connected by the formulas* (9), (10), (11). *Here* r_σ *and* q_ρ *can be defined in the following manner:* $\mathfrak{F}_\sigma$ *represents a complete matric algebra over a Galois field of degree* $\mathfrak{r}_\sigma$ *over* $\bar{\mathfrak{o}} \cong \mathfrak{o}/\mathfrak{p}$ *and* $\mathfrak{T}_\rho$ *represents a complete matric algebra over a division algebra of rank* $\mathfrak{q}_\rho$ *over* $K_\mathfrak{p}$.

By means of (9), (10), (11) we can compute the multiplicity $c_{\kappa\lambda}/r_\lambda$ of $\mathfrak{F}_\lambda$ in $\mathfrak{U}_\kappa$. We thus find

$$c_{\kappa\lambda} = \sum_{\rho=1}^{l} d_{\rho\kappa} d_{\rho\lambda} q_\rho \qquad (\kappa, \lambda = 1, 2, \ldots, k), \tag{12}$$

a formula which expresses the Cartan invariants $c_{\kappa\lambda}$ of $J/\mathfrak{p}$ by means of the "*decomposition numbers*" $d_{\rho\kappa}$. In particular, $c_{\kappa\lambda} = c_{\lambda\kappa}$. It may be remarked that $J/\mathfrak{p}$ need not be a symmetric algebra. In the case of a group ring, we obtain therefore an "arithmetical" proof for the symmetry of the Cartan invariants, which is quite different from the "algebraic" proof given in B. N.

We may apply similar arguments in the case of the indecomposable constituents $\mathfrak{V}_\sigma$ of the second regular representations $\mathfrak{S}$ of $J/\mathfrak{p}$. We have formulas analogous to (9) and (11). Comparing them with (9) and (11), we find

$$(\mathfrak{V}_\sigma) \longleftrightarrow \sum_{\rho=1}^{l} d_{\rho\sigma}\,\mathfrak{T}_\rho \longleftrightarrow (\mathfrak{U}_\sigma). \tag{13}$$

If A is normal and simple, we have only one $\mathfrak{T}_\lambda$, $l = 1$, and the corresponding q_λ gives the square of the $\mathfrak{p}$-index of A.[5]

In the case of a semisimple algebra A, we can always replace K by an algebraic extension field such that with regard to this new ground field, the numbers q_ρ and r_σ are to be replaced by 1. With regard to such a ground field, we have

$$(\mathfrak{U}_\sigma) \longleftrightarrow (\mathfrak{V}_\sigma) \longleftrightarrow \sum_{\rho} d_{\rho\sigma}\mathfrak{T}_\rho;\ \mathfrak{T}_\rho \longleftrightarrow \sum_{\sigma} d_{\rho\sigma}\,\mathfrak{F}_\sigma, \tag{14}$$

$$c_{\kappa\lambda} = \sum_{\rho} d_{\rho\kappa}d_{\rho\lambda}. \tag{15}$$

[1] For the properties of the regular representations and intertwining matrices cf. R. Brauer–C. Nesbitt, these PROCEEDINGS **23**, 236 (1937) (referred to under B. N.). T. Nakayama, *Ann. of Math.* (2), **39**, 361 (1938); C. Nesbitt, *Ann. of Math.* (2), **39**, 634 (1938); and two forthcoming papers by R. Brauer.

[2] See L. E. Dickson, *Algebras and Their Arithmetics*, Chicago, 1923, §42.

[3] Cf. for instance, A. A. Albert, *Modern Higher Algebra*, Chicago, 1937, p. 296.

[4] W. Burnside, *Proc. London Math. Soc.* (2), **7**, 8 (1909).

[5] H. Hasse, *Math. Ann.*, **107**, 731 (1933).

NUMBER OF THE SUBGROUPS OF ANY GIVEN ABELIAN GROUP

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Every abelian group whose order is not a power of a prime number is known to be the direct product of its Sylow subgroups. Hence the number of its subgroups is the product of the numbers of the subgroups contained in its Sylow subgroups. It is therefore easy to determine the total number of the subgroups of any given abelian group whenever we know the numbers of the subgroups contained in its Sylow subgroups. Hence we shall confine our attention for the present to a determination of the total number of the subgroups contained in a given abelian group G of order p^m, p

being a prime number. It is known that every possible set of independent generators of G involves the same number of operators. Let $s_1, s_2, \ldots, s_k$ be such a set of independent generators of G, arranged in ascending order of magnitude if the orders of two such successive generators are not all equal to each other.

We shall first determine the total number of the cyclic subgroups of an arbitrary order p^α contained in G. For this purpose it is only necessary to find the total number of the operators of G whose orders divide p^α and to subtract from this number the total number of the operators of G whose orders are less than p^α. The remainder thus obtained divided by $p^\alpha - p^{\alpha-1}$ gives the total number of the cyclic subgroups of order p^α contained in G since each of these cyclic subgroups contains exactly $p^\alpha - p^{\alpha-1}$ operators of order p^α which do not appear in any other cyclic group of order p^α, because these operators separately generate the cyclic group of this order in which they appear.

The number of the operators contained in G whose orders divide p^α is the product of k numbers, r of these are equal to p^α and represent respectively orders of operators generated by the given set of independent generators of G whose orders are separately at least equal to p^α, while the remaining $k - r$ are respectively equal to the orders of the operators in the given set of independent generators which are separately less than p^α. The number of the operators contained in G whose orders are less than p^α is also equal to the product of k numbers. The last $k - r$ of these may be so selected that they are the same as in the preceding case, while the remaining r of them are separately equal to $p^{\alpha-1}$ instead of p^α. Hence there results the following theorem: *If an abelian group of order* p^m, p *being a prime number, has a set of* k *independent generators which involves* r *operators which are separately at least equal to* p^α *while the orders of the rest of these operators are separately less than* p^α, *the number of its cyclic subgroups of order* p^α *may be obtained by dividing by* $\mathrm{p} - 1$ *the product of the latter orders and* $(\mathrm{p}^r - 1)\mathrm{p}^{(\alpha-1)(r-1)}$.

The number of the operators of order p^α contained in G is always larger than the number of its cyclic subgroups of this order except when $p = 2$ and $\alpha = 1$. In this special case these two numbers are equal to each other. If the order of an abelian group is not a power of a prime number its cyclic subgroups are the direct products of the cyclic subgroups contained in its Sylow subgroups. Hence the number of its cyclic subgroups is then the product of the numbers of its cyclic subgroups contained in its Sylow subgroups. It may be noted here that in the *Encyclopaedia Britannica* (1938) there appears a statement under the entry of the term "Groups," volume 10, page 914, which implies that a set of independent generators of an arbitrary abelian group can be so selected that each of them generates a Sylow subgroup. This is evidently only possible when the given abelian

group is cyclic. Various other incorrect statements appear under the same entry in this encyclopedia.

It is considerably more difficult to determine the number of the non-cyclic subgroups of G than to find a formula which gives the number of the cyclic subgroups contained therein. A few general theorems simplify the determination of the former number. One of these may be stated as follows: An abelian group of order p^m contains one and only one subgroup which satisfies the two conditions that it has a set of independent generators involving as many operators as appear in a set of independent generators of G and that the orders of the former independent generators are all equal to a given power of p which does not exceed the order of the smallest independent generator of G. The number of these subgroups contained in G is therefore equal to the index of the power of p which is equal to the order of the smallest independent generator of G. Each of these subgroups is a characteristic subgroup of G since it is composed of all the operators of G whose orders divide a given number.

Another general theorem relating to the number of the subgroups of G may be stated as follows: If all the orders of a set of independent generators of a subgroup of G are equal to the orders of a set of independent generators of G with the exception of the generator of highest order in the latter set, and the order of the largest independent generator of the subgroup is at least equal to the order of the next to the largest independent generator of G, then there is one and only one such subgroup in G for every order of the last independent generator of the subgroup which does not exceed the order of the largest independent generator of G. The number of these subgroups of G is therefore equal to one more than the difference between the indexes of the power of p which is equal to the order of the largest independent generator of G and the power of p which is equal to the order of the next to the largest operator in the given set of the independent generators of G.

Since the number of the operators in a set of independent generators of a subgroup of G is equal to the index of the power of p which is equal to the order of the group generated by all the operators of order p contained in this subgroup it results that a subgroup of G cannot have a set of independent generators which involves more operators than appear in every set of independent generators of G. In particular, a necessary and sufficient condition that a subgroup of G has a set of as many independent generators as G itself has, is that it involves all the operators of order p contained in G. To determine the number of the subgroups of a given type which appear in G it is only necessary to determine the number of ways in which a set of independent generators of a subgroup of this type can be selected from the operators of the group and to divide this number by the number of ways in which a set of independent generators of such a subgroup can be selected

from its own operators. In particular, the number of the subgroups of G which have $k_1 \leqq k$ independent generators and involve no operator of order p^2 is known to be

$$\frac{(p^k - 1)(p^k - p)\ldots(p^k - {k_1}^{k-1})}{({k_1}^k - 1)({k_1}^k - p)\ldots({k_1}^k - {k_1}^{k-1})}.$$

In determining the number of the subgroups of a given type contained in G it is only necessary to consider the operators of G whose orders do not exceed the order of the largest operator in a set of independent generators of such a subgroup. It is convenient to consider first the number of ways in which an independent generator of highest order of such a subgroup can be selected from the operators of G and then to consider the number of ways in which the independent generators of the successively lower orders in a set of independent generators of the subgroup can be selected from the operators of G and to divide the product of these numbers by the number of ways in which the operators of a set of independent generators of the subgroup can be selected from the operators of the subgroup. In each particular case it is only necessary to consider the operators of the group, or of the subgroup, whose order is equal to the order of the independent generator in question.

To illustrate this general method we proceed to consider the total number of the subgroups of G when $k = 2$ and the orders of s_1 and s_2 are p^β and p^γ, respectively. When the smaller of the two generators of such a subgroup is p the larger independent generator can be selected from the operators of G in $p^{\beta-1}$ times as many ways as from the operators of the subgroup while the smaller of the two independent generators can then be selected from the operators of G in the same number of ways as from the operators of the subgroup in question provided the order of the larger independent generator of the subgroup is at least p^β. As the order of the larger independent can be chosen in $\gamma - \beta$ ways so as to exceed p^β the number of these subgroups in which one independent generator is of order p is $(\gamma - \beta)p^{\beta-1}$. As similar remarks apply to the cases when the smaller independent generators of the subgroup in question have various larger values up to p^β, the number of the subgroups in which the larger independent generator is at least as large as p^β is $(\gamma - \beta)(p^\beta - 1)/p - 1$.

We proceed to consider the case when neither of the two generators of the subgroups in question has an order which exceeds p^β. If these generators have the same order there is clearly one and only one subgroup since such a subgroup is then composed of all the operators of G whose orders divide the common order of the generators. It remains to consider the case when the two independent generators of the subgroups in question have different orders, say p^{α_1}, p^{α_2}, $\alpha_2 > \alpha_1$. The number of the operators of order p^{α_2} contained in G is then $p^{2\alpha_2} - p^{2\alpha_2-2}$ and the number of the

operators of this order contained in such a subgroup is $p^{\alpha_1+\alpha_2} - p^{\alpha_1+\alpha_2-1}$. The number of these subgroups is therefore the quotient of the given numbers of operators since the numbers of ways in which the smaller independent generator can be chosen after the larger independent generator has been selected is the same for the group as for the subgroup.

Hence the number of the subgroups in which the two independent generators are of orders p^{α_1} and p^{α_2} is $p + 1$ times $p^{\alpha_2-\alpha_1-1}$. The exponent $\alpha_2 - \alpha_1 - 1$ cannot exceed $\beta - 2$ and has this value only when $\alpha_2 = \beta$ and $\alpha_1 = 1$. There are two cases in which this exponent is $\beta - 3$, viz., when $\alpha_2 = \beta$ and $\alpha_1 = 2$ or when $\alpha_2 = \beta - 1$ and $\alpha_1 = 1$. As similar considerations apply to the other cases the number of these subgroups is obviously given by the formula

$$(p + 1)(p^{\beta-2} + 2p^{\beta-3} + \ldots\ldots + (\beta - 1)p^0$$

whenever $\beta > 1$. In this case the total number of the subgroups in the abelian group of order p^m whose independent generators are of orders p^β, p^γ is therefore given by the formula

$$\beta + (p + 1)(p^{\beta-2} + 2p^{\beta-3} + \ldots\ldots + (\beta - 1)p^0) + (\gamma - \beta)\,(p^\beta - 1)/(p - 1).$$

The entire group is included among the subgroups in the enumeration by this formula. When $\beta = 1$ the first and the last term of this formula, taken together, give the total number of the subgroups in question.

THE TRANSFORMATION OF A LAGRANGIAN SERIES INTO A NEWTONIAN SERIES

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A series of type

$$\sum_{n=0}^{\infty} a_n \frac{(z - c - 1)(z - c - 2) \ldots (z - c - n)}{(z + c + 1)(z + c + 2) \ldots (z + c + n)} = \sum a_n R_n(z) \quad (1)$$

will be called a Lagrangian series on account of the extensive investigations of René Lagrange[1] relating to the expansion of functions in series of this and allied types.

A transformation of a Newtonian series into a series of type (1) may be effected with the aid of Lagrange's expansion

$$p(z + c/, p) = \Sigma\,(2c + 2n + 1)(n + p - 1/, p - 1)\times (2c + n/, p - 1)R_n(z) \quad (2)$$

where the symbol $(q/, p)$ is used for the coefficient of t^p in $(1 + t)^q$. When $c = 0$ the transformation of the series (1) into a Newtonian series may be based on the formula

$$\frac{(1 - z, p)}{(1 + z, p)} = \sum_{n=p+1}^{\infty} (-)^{n+p-1}\,(z/,n)\,\frac{(1 - n, p)}{(1 + n, p)} \quad (3)$$

where $(m, n) = m(m + 1)(m + 2) \ldots (m + n - 1)$, $(m, 0) = 1$, $(m, 1) = m$.

To prove this formula we observe that the series on the right may be written in the form

$$z(1 - z)(2 - z) \ldots (p - z)p\,F(p + 1, 1 + p - z;\ 2p + 2;\ 1)/(2p + 1)! \quad (4)$$

When $R(z) > 0$ the hypergeometric series may be summed by the formula of Gauss and has the value

$$(2p + 1)!\ /z(z + 1)(z + 2) \ldots (z + p)p!$$

We thus obtain the expression on the left.

The relation (3) gives rise to a number of interesting expansions of which we mention three

$$F(1 - z;\ 1 + z;\ x) = \sum_{n=1}^{\infty} (-)^{n-1}\,(z/,n)F(1 - n;\ 1 + n;\ -x)$$

$$F(a, 1 - z;\ 1 + z;\ x) = \sum_{n=1}^{\infty} (-)^{n-1}\,(z/,n)F(a, 1 - n;\ 1 + n;\ -x) \quad (5)$$

$$F(1 - z;\ 1 + z, a;\ x) = \sum_{n=1}^{\infty} (-)^{n-1}\,(z/,n)F(1 - n;\ 1 + n, a;\ -x).$$

In the second of these we suppose that $R(a)$ is negative when $x = -1$; otherwise we must suppose that $|x| < 1$. In the former case we may use Gauss's formula for the hypergeometric series and obtain the identity

$$F(a, 1 - z;\ 1 + z;\ -1) = zF(1 - z, 1 - {}^1/_2a, {}^3/_2 - {}^1/_2a;\ {}^3/_2, 2 - a;\ 1) \quad R(a) < 0 \quad (6)$$

which is certainly true when $a = -1$. It is also true when $a = 0$; When $c = 0$ the expansion of a function $f(z)$ in a series of type (1) may be written in the symbolical form

$$f(z) = \frac{1}{z} f(1) + \sum_{n=1}^{\infty} (-)^n \frac{(z-1)(z-2) \ldots (z-n)}{z(z+1)(z+2) \ldots (z+n)} \times (2n+1) P_n(1-2E) f(1) \quad (7)$$

where E^s is an operator which changes $f(1)$ into $f(s+1)$. When the operations are performed the series gives a formula of interpolation. A formula something like this has been mentioned by E. Hille.[2] When $f(z)$ is a function represented by a Laplacian integral

$$f(z) = \int_0^{\infty} e^{-zt} g(t) dt \quad (8)$$

in which $g(t)$ is continuous we obtain a confirmation of Lerch's result[3] that when $F(1), f(2), f(3) \ldots$ are all zero $f(z)$ is zero for all values of z exceeding unity.

An interesting example of the formula of interpolation (7) is furnished by the equation

$$\frac{uz}{u+z-1} = F(1, {}^3/_2, 1-u, 1-z; {}^1/_2, 1+u, 1+z; 1) \quad (9)$$

the generalized hypergeometric series being convergent when $R(u+z) > 1$. Converting this series into a Newtonian series by means of the relation (3) we obtain the equation

$$\frac{uz}{u+z-1} = \sum_{n=1}^{\infty} (-)^{n-1} (z/, n) F(1, {}^3/_2, 1-u, 1-n; {}^1/_2, 1+u, 1+n; -1). \quad (10)$$

Calculating the coefficients in the Newtonian series by the usual rule we find that

$$F(1, {}^3/_2, 1-u, 1-n; {}^1/_2, 1+u, 1+n; -1) = n!/(u+1, n-1). \quad (11)$$

The hypergeometric series on the left is "nearly-poised" in the terminology of F. J. Whipple.[4] The identity (6) is a particular case of Whipple's identity

$$\Gamma({}^1/_2 k - b)\, \Gamma(k)\, F(a, b; k-b; -1) = \Gamma(k-b)\, \Gamma({}^1/_2 k) F(b, {}^1/_2 k - {}^1/_2 a, {}^1/_2 + {}^1/_2 k - {}^1/_2 a; k-a, {}^1/_2 + {}^1/_2 k; 1) \quad (12)$$

which is an extension of one due to Kummer. The relation (11) is a particular case of Whipple's identity[5] $F(a, 1 + {}^1/_2 a, c, d; {}^1/_2 a, 1+a-c, 1+a-d; -1) = F(c, d; 1+a; 1)$.

Another interesting example of the formula (7) is obtained by writing (9) in the form

$$1/(u + z - 1) = \sum_{n=0}^{\infty} (2n + 1)G_n(u)G_n(z), \tag{13}$$

where $z(1 + z, n)G_n(z) = (1 - z, n)$.

With this notation the Legendre series associated with $(1/2 - 1/2s)^{z-1}$ is

$$(1/2 - 1/2s)^{z-1} \sim \sum_{n=0}^{\infty} (2n + 1)P_n(s)G_n(z) \tag{14}$$

and a related series is

$$\int_0^{\infty} e^{-xt} (1 + t)^{-z} dt = \sum_{n=0}^{\infty} (2n + 1) V_n(x)G_n(z), \tag{15}$$

where $V_n(x) = \int_0^{\infty} e^{-xt} P_n[(t - 1)/(t + 1)]dt/(t + 1)$.

If $R(z) > 0$ there is a further relation

$$t^{z-1} / \Gamma(z) = \sum_{n=0}^{\infty} (2n + 1)Z_n(t)G_n(z), \tag{16}$$

where $Z_1(t) = F(-n, n + 1; 1, 1; z)$. Also, if $R(z) > 1$, $0 < u \leq 1$

$$\Gamma(u + z - 1)/\Gamma(u)\,\Gamma(z) = \sum_{n=0}^{\infty} (2n + 1)F_n(2u - 1)G_n(z)$$

where $F_n(2u - 1) = F(-n, n + 1, u; 1, 1; 1)$.

[1] R. Lagrange, *Acta Mathematica*, **64**, 1–80 (1935).
[2] E. Hille, *Compositio Mathematica*, **6**, 93–102 (1938).
[3] M. Lerch, *Acta Mathematica*, **27** (1903).
[4] F. J. Whipple, *Proc. Lond. Math. Soc.* (2), **26**, 257–272 (1927).
[5] F. J. Whipple, *Ibid.* (2), **24**, 247–263 (1925).

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